JOURNAL OF INONU LIVER TRANSPLANTATION INSTITUTE

DOI: 10.14744/jilti.2025.18209
J Inonu Liver Transpl Inst 2024;2(3):135–138

Case Report

Coma Blister Mimicking Necrotizing Fasciitis in a Liver Transplant Patient: A Case Report

Adem Tuncer,[1] Mehmet Zeki Ogut,[2] Sertac Usta,[3] Fatih Ozdemir,[3] Sezai Yilmaz[3]

[1]Department of General Surgery, İstanbul Aydın University, İstanbul, Türkiye
[2]Department of General Surgery, Elazığ Fethi Sekin City Hospital, Elazığ, Türkiye
[3]Department of General Surgery and Liver Transplantation Institute, Inonu University Faculty of Medicine, Malatya, Türkiye

Abstract

Coma blisters (CB) are self-limiting cutaneous lesions that typically occur in patients with prolonged impaired consciousness, often due to drug overdoses such as barbiturates. They are rarely observed in liver transplant patients and can clinically mimic conditions like necrotizing fasciitis. We report the case of a 49-year-old male post-liver transplant who presented with bullous and necrotic lesions on the anterior abdominal wall, initially suspected to be necrotizing fasciitis. A skin biopsy confirmed CB, and the patient responded to corticosteroid treatment, with improvement over 20 days. The patient returned 10 months later with similar lesions, which again resolved with similar management. Early diagnosis and appropriate treatment are essential, particularly in immunocompromised patients, to distinguish CB from more serious conditions.

Keywords: Coma blisters, necrotizing fasciitis, liver transplantation

Please cite this article as "Tuncer A, Ogut MZ, Usta S, Ozdemir F, Yilmaz S. Coma Blister Mimicking Necrotizing Fasciitis in a Liver Transplant Patient: A Case Report. J Inonu Liver Transpl Inst 2024;2(3):135–138".

Coma blister (CB) is usually seen as a result of the overdose of drugs such as benzodiazepines, antipsychotics, opiates, tricyclic antidepressants, alcohol, heroin, and barbiturates. It is rarely seen in patients with long-term impaired consciousness.[1,2] It has also been observed in patients with chronic renal failure, hypercalcemia, carbon monoxide poisoning, diabetic ketoacidosis, and various neurological disorders.[3] Skin lesions usually appear after 48-72 hours and resolve within 2-4 weeks.[1,2] The mechanism of CB formation is not fully understood, but timely treatment is essential. CB is a self-limiting cutaneous disease. Patients may present with complaints of pain, edema and skin necrosis in the affected area. Because it is rarely seen, clinicians may it misdiagnose as necrotizing fasciitis or cellulitis.[4] A 47-year-old male patient presented with the complaint of extensive skin necrosis on the anterior abdominal wall. We present the case because this situation is special in a patient who had a liver transplant before.

Case Report

A 49-year-old male patient who had undergone liver transplantation for Budd-Chiari 5 years ago was continuing his routine controls. Three years after liver transplantation, the patient was admitted to our clinic with ecchymosis, bullous and necrotic lesions on the anterior abdominal wall for 3 days (Fig. 1). On the physical examination, necrotizing fasciitis was considered in the differential diagnosis, and antibiotic therapy was started since there was ecchymosis in an area localized with bullae on the right side of the anterior abdominal wall and severe pain on palpation. Neurological

Address for correspondence: Adem Tuncer, MD. Department of General Surgery, İstanbul Aydın University, İstanbul, Türkiye
Phone: +90 537 564 83 18 **E-mail:** ademtuncer89@hotmail.com

Submitted Date: 09.12.2024 **Revised Date:** 02.01.2025 **Accepted Date:** 03.01.2025 **Available Online Date:** 24.01.2025

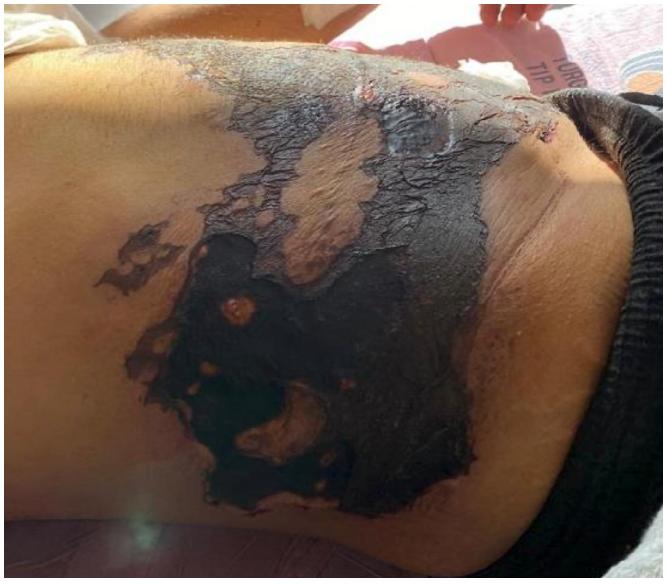

Figure 1. Picture of the patient's lesion at the time of admission.

examination of the patient with high acute phase reactant levels was normal.

There was no finding in favor of necrotizing fasciitis in the abdominal tomography of the patient (Fig. 2). The patient who had a liver transplant due to Budd-Chiari was regularly using tacrolimus, warfarin, ursodeoxycholic acid, and esomeprazole. The patient's tacrolimus level was normal (7.6: 5-20), and his INR was 1.6. A biopsy was taken from the patient's lesion and the patient was consulted with the dermatology department. Purpura fulminans, warfarin necrosis, bullous pyoderma gangrenosum, ecthyma gangrenosum, necrotizing fasciitis were considered in the differential diagnosis. In addition to antibiotics, methylprednisolone 40 mg and the wet dressing were started as treatment. In addition, the immunosuppression dose taken by the patient was decreased and continued.

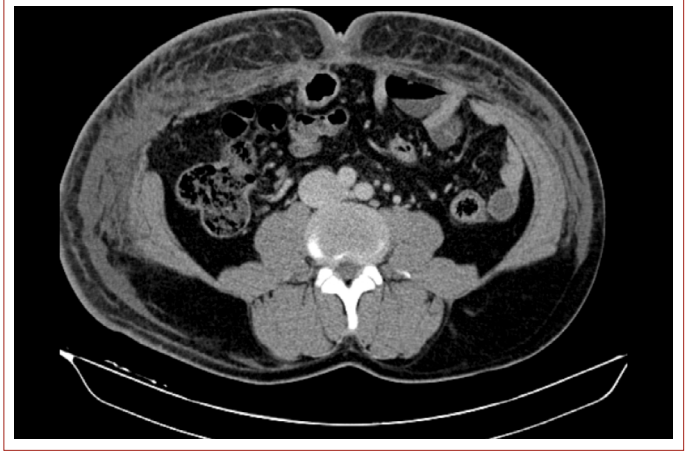

Figure 2. Abdominal tomography of the patient at the time of admission.

The patient's complaints regressed after 7 days and the color of the necrotic areas began to improve (Fig. 3). Heparin treatment was started for the patient using Coumadin. And the dose of methylprednisolone was gradually reduced. The wound dressing was continued with antibiotic creams. After 20 days of hospitalization, necrotic tissues were debrided and wound dressing of the ulcerated area was continued. After 4 weeks, the pathology result was reported as a coma blister. And the patient's methylprednisolone and the antibiotic cream dressing were continued. After 7 days, the methylprednisolone treatment was stopped. The patient was discharged on the 40th day.

10 months later, the patient came back to the clinic with complaints of redness, bruising and pain in the right thigh (Fig. 4). The necrotic lesion, which was severely painful on palpation, resembled the lesions 10 months ago. The patient was hospitalized and after 1 week of antibiotic, steroid and anticoagulant treatment, the lesion resolved and the patient was discharged. No new lesion developed in the follow-up of the patient (Fig. 5).

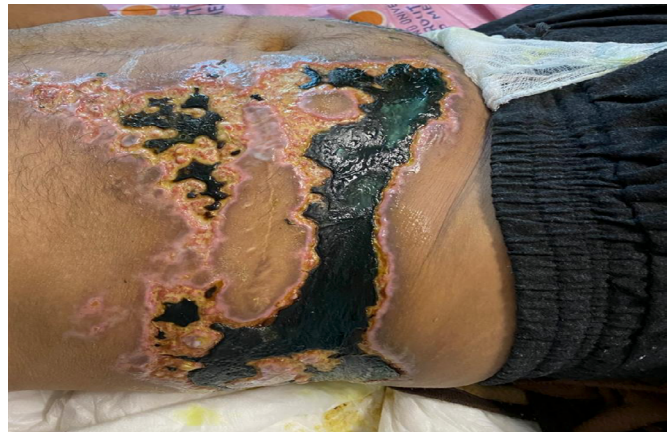

Figure 3. Picture of the patient's lesion during treatment.

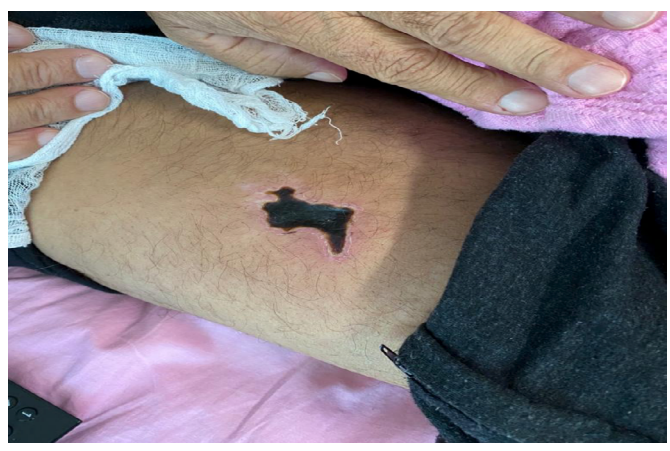

Figure 4. Picture of the lesion developing on the patient's thigh.

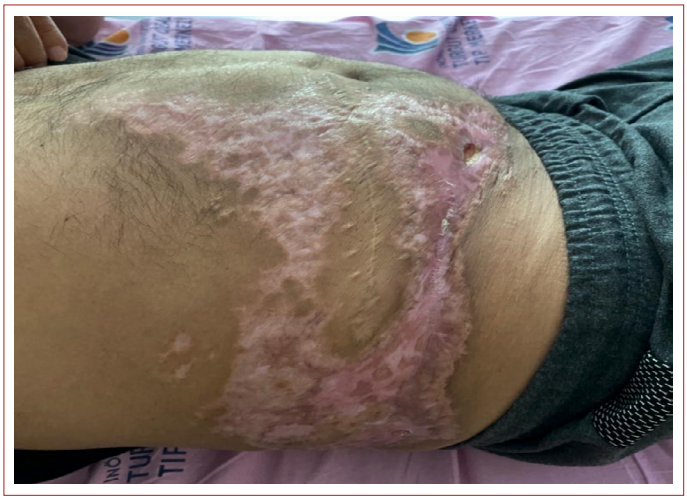

Figure 5. Picture of patient's response to treatment 6 months after treatment.

Discussion

Coma blisters are self-limiting lesions that occur in a comatose state for a variety of reasons but are most commonly associated with barbiturate overdose. Examination of the skin biopsy specimen shows the characteristic presence of eccrine sweat duct necrosis.[5] When CB was first described in the medical literature in 1965, it was reported that CBs were caused by barbiturate poisoning.[6] Later, it was thought that CB was caused by pressure in patients with impaired consciousness and ischemia in places where the pressure was high. However, due to its distribution and localization, this situation could not be explained by increased pressure or vascular pathology. While it has been suggested that some toxic effects of drugs may play a role, the relationship between CB and any specific drug has not been proven. He demonstrated that these skin lesions caused by barbiturates can be distinguished by the specific histological finding of sweat gland necrosis. They can usually be seen in both pressurized and non-pressurized areas on the extremities and trunk.[7]

There are also studies reporting damage to sweat glands and ducts and related tissue hypoxia as the main mechanism of CBs.[8] CB is located on pressure areas, suggesting that dermal pressure injury is an important underlying mechanism. However, they have also been reported to affect non-pressure sites, suggesting that additional mechanisms may be involved, including drug toxicity, hypoxia, hypothermia, metabolic acidosis, and immune system disorders. In the differential diagnosis, pemphigoids may include epidermolysis bullosa, bullous drug eruptions, bullous diabeticorum and postburn bullae.[2]

CB, which is not well defined in the literature, should be suspected in patients with severe pain, as it may cause irreversible nerve and muscle damage.[4] Necrotizing fasciitis is a serious, potentially fatal soft tissue infection that progresses rapidly and can develop into septic shock.[9] 70-90% of these patients are polymicrobial and they are accompanied by various comorbidities such as diabetes mellitus.[10] These two diseases can mimic each other. The etiology, clinic and treatment of coma blister have been discussed above. Necrotizing fasciitis is a life-threatening bacterial infection that rapidly destroys subcutaneous tissue that reaches the muscles. While coma blister is usually painless and can be healed with simple wound care, necrotizing fasciitis causes severe pain and requires surgical debridement and broad-spectrum antibiotic therapy. If not treated early, necrotizing fasciitis can lead to serious consequences such as sepsis, organ loss and death, while coma blister usually does not cause such complications.[11]

A biopsy was taken from the lesion that clinically and radiologically mimicked necrotizing fasciitis in our patient, and symptomatic treatment was started. As a result of the skin biopsy, the diagnosis of CB was confirmed.

Because CB lesions are self-limiting, a broad-spectrum topical antibiotic cream is sufficient to prevent secondary infection of ruptured bullae. Most bullae heal within two weeks with the regeneration of clinically intact normal skin.[7]

Conclusion

Coma blisters are lesions that can accompany various neurological diseases. Although they are most commonly associated with barbiturate overdose, they can also be seen in coma due to other etiologies. Blisters develop 48 to 72 hours after the onset of unconsciousness. Because liver transplant patients are immunosuppressed, early diagnosis and treatment for coma blisters are important.

Disclosures

Informed Consent: Written informed consent was obtained from the patient for the publication of the case report and the accompanying images.

Peer-review: Externally peer-reviewed.

Conflict of Interest: None declared.

Authorship Contributions: Concept – A.T., S.U.; Design – A.T., S.U., F.O.; Supervision – A.T., S.U., F.O.; Materials – A.T., Z.O., S.U., F.O.; Data collection &/or processing – A.T., Z.O.; Analysis and/or interpretation – A.T., Z.O., S.U., F.O.; Literature search –A.T., Z.O., S.U.; Writing – A.T., Z.O., S.U; Critical review – A.T., S.U.

References

1. Chacon AH, Farooq U, Choudhary S, Yin N, Nolan B, Shiman M, et al. Coma blisters in two postoperative patients. Am J Dermatopathol 2013;35(3):381–4.

2. Bosco L, Schena D, Colato C, Biban P, Girolomoni G. Coma blisters in children: case report and review of the literature. J Child Neurol 2013;28(12):1677–80.
3. Piede J, Wallace E. Coma bullae: Associations beyond medications. Mayo Clin Proc 2011;86(1):4065.
4. Ruiz-Rivero J, Pulido-Pérez A, Suárez-Fernández RM. Coma blisters with deep soft tissue involvement after drug overdose. Int J Dermatol 2017;56(8):881–3.
5. Dunn C, Held JL, Spitz J, Silver DN, Grossman ME, Kohn SR. Coma blisters: report and review. Cutis.1990 ;45(6):423–6.
6. Beveridge GW, Lawson AAH. Occurrence of bullous lesıons ın acute barbıturate ıntoxıcatıon. Br Med J 1965;1(5438):824–37.
7. Keng M, Lagos M, Liepman MR, Trever K. Phenobarbital-Induced Bullous Lesions in a Non-Comatose Patient. Psychiatry (Edgmont) 2006;3(12):65.
8. Miyamoto T, Ikehara A, Kobayashi T, Kitada S, Hagari Y, Mihara M. Cutaneous eruptions in coma patients with nontraumatic rhabdomyolysis. Dermatology 2001;203(3):233–7.
9. Misiakos EP, Bagias G, Patapis P, Sotiropoulos D, Kanavidis P, Machairas A. Current concepts in the management of necrotizing fasciitis. Front Surg. 2014 Sep 29;1.
10. Hakkarainen TW, Kopari NM, Pham TN, Evans HL. Necrotizing soft tissue infections: Review and current concepts in treatment, systems of care, and outcomes. Curr Probl Surg 2014;51(8):344.
11. Chen LL, Fasolka B, Treacy C. Necrotizing fasciitis: A comprehensive review. Nursing 2020;50(9):34-40.